INFANT ACCEPTANCE OF BREAST MILK FOLLOWING SEVERE MATERNAL EXERCISE

THESIS
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INTRODUCTION AND
AIM OF THE WORK
INTRODUCTION

Some lactating women report difficulty nursing their infants following exercise. These mothers note that their infants refuse to nurse or fuss during the post exercise feeding. In a survey of lactating women who exercise at a moderate intensity 7% reported that their infants “often” had difficulty nursing following maternal exercise (Brown and Wallace, 1990).

It was postulated that there may be some byproduct of exercise that may affect the milk. In pervious studies it was found a significant increase in lactic acid concentration in breast milk following maximal exercise (Wallace and Rabin, 1991).

Lactic acid produces a sour taste that may be detected by the infant Guyton (1981). Steiner (1977) documented a gustofacial response (puckering facial expression) to sour taste in infants as early as a few hours after birth it is described as a low level reflex not involving cortical structures.

Engen (1977) demonstrated a more rapid rate of suckling to a sweet taste than to a bitter taste.

Jonson and Salisbury (1977) observed changes in heart rate, respiratory rate and suckling pattern when infants were presented with unfamiliar milk.
Infants sense sweet and sour tastes. Taste buds, found to be at the biggest number in the 5 to 7 months old fetus and reach adult morphology at 13 to 15 weeks in utero and are complete before birth (Farbman, 1971).

AIM OF THE WORK

The aim of this study is to compare infant acceptance of pre-exercise and post-exercise breast milk and to correlate the infant response to the concentration of lactic acid in the milk.
REVIEW OF LITERATURE
HUMAN BREAST MILK

Breast milk is a natural milk, species specific, the differential constituents of milk differ in order to fulfill the requests of each newborn (Pereira and Barbosa 1986).

The human breast milk guarantees the optimum nutrient for the newborn irrespective of gestational age being the most suitable milk for fullterm, preterm and even risk babies (Anderson, 1985).

Breast feeding continuous to have practical and psychological advantages that should be considered when the mother selects the method for feeding. Human milk is the most appropriate of all available milks for the human infant because it is uniquely adapted to his or her needs (Barness, 1996).
ANATOMY OF THE HUMAN BREAST

The mammary gland of the human female consists of glandular epithelium and duct system embedded in interstitial tissue and fat. Human breast is composed of 15 to 20 tubulo-alveolar glands embedded in fat and open into lactiferous ducts, which ends as small orifices near the tip of the nipple (*Jelliffe and Jelliffe, 1979*).

The center of fully developed breast in adult woman is marked by the areola which is a circular pigmented skin surface appears rough because of the presence of large modified sebaceous glands called the tubercle of Montgomery which provide nipple lubrication and antisepsis. The nipple contains nerve fibers, muscle fibers and sweat glands as well as mild duct openings. The major blood supply to the breast is from branches of intercostal arteries and internal thoracic artery. Sensory and sympathetic fibers from the fourth, fifth and sixth intercostal nerve innervate the breast. The nipple and areola have more extensive innervation than the glandular tissue (*Lawrence, 1985*).

The alveoli of the lactating gland are composed of cuboidal epithelial and myoepithelial cells. During the first trimester of pregnancy, under influence of circulating hormones, rapid growth occurs and the tubulo alveolar portions of glands develop. During pregnancy and lactation breast size increases from approximately 200 gm to 600-800 gm in the mature breast (*Lawrence, 1985*).
PHYSIOLOGY OF LACTATION

Breast feeding process results from the interplay of hormones, initiative reflexes and learned behaviour by mother and newborn (Kon, 1972).

Breast feeding depends on the physiological process:
I- Mamogenesis:
Mammary growth takes place in four different stages:
a- Pr-pupertal growth:
The primary and secondary ducts that develop in the fetus in utero continue to grow in both male and female in proportion to growth in general. Shortly before puberty, a more rapid expansion of ducts begins in the female depending on estrogen. Such expansion doesn't occur in absence of ovaries. The complete growth of alveoli requires stimulation by progesterone as well (Lawrence, 1985). It has been shown that the secretion of prolactin and somatotropines hormones and thyroid stimulating hormone acting a role in growth of the mammary gland Cowie, (1972).

b- Pupertal growth:
When the hypophyseal ovarian uterine cycle is established there is extensive branching of the duct system and proliferation and canalization of lobulo-alveolar units at the distal tips of the branches (Lawrence, 1985).
c- Menstrual cycle growth:

The cyclical changes of the mammary gland associated with the menstrual cycle are due to the hormonal changes that control the cycle. Estrogen stimulates parenchymal proliferation with formation of epithelial sprouts. This hyperplasia is continuous into the secretory phase of the cycle. When corpus luteum provides increased amount of estrogen and progesterone, there is increased mammary blood flow in the luteal phase. The ovulatory cycle actually enhances mammary growth until about age of thirty (Lawrence, 1985).

d- Mammary growth in pregnancy:

The principle feature of mammary growth in pregnancy is a great increase in duct and alveoli, under the influence of many hormones including estrogen, progesterone, corticosteroids and hypophyseal and placental lactogens. Nipple length and protractility also increase (Jelliffe and Jelliffe, 1979).

Placenta has been found to play an important role in mammary growth. In pregnancy the placenta secretes ovarian like hormones in large quantities (Lawrence, 1985). Late in pregnancy there is a maximal development of the lobulo-alveolar system and sensitization of glandular tissue for the action of prolactin. Also, colostrum is secreted in small amount during the last three months of pregnancy (Jelliffe and Jelliffe, 1979).
(II) Lactogenesis or initiation of milk secretion:

Three main hormones are recognized in the lactogenic process prolactin hormone, human placental lactogen and human growth hormone. (Lawrence, 1985). Initiation of lactogenesis is closely related to the rising levels of prolactin, which enhance specific enzymes concerned with synthesis of milk protein and lactose (Hambraeus et al., 1984).

In addition estrogen, progesterone, hydrocortisone and insulin promote further development of glandular component of breast. Prolactin is a significant hormone in pregnancy and lactation (Frantz, 1978).

Prolactin level is the same in both human male and female until puberty. At puberty, the increased estrogen in female causes a measurable increase in prolactin. Also during the proliferative phase of menstrual cycle there is increase in prolactin level. (Josimovich et al., 1974). In pregnancy, prolactin level begins to rise in the first trimester and continue to rise throughout gestation. At delivery with expulsion of placenta there is abrupt decline in estrogen and progesterone. Estrogen enhances the effect of prolactin on mammogenesis but antagonises prolactin on milk secretion thus withdrawal of estrogen after expulsion of placenta triggers the onset of lactation (Lawrence, 1985).

Suckling stimulates the release of adenohypophyseal prolactin and neurohypophyseal oxytocin. These hormones stimulate milk
synthesis and ejection. The most effective and specific stimulus to prolactin release is nursing Lawrence (1985).

Prolactin releases from the anterior pituitary and is inhibited by prolactin inhibiting factor released from the hypothalamus which is mediated by the catecholamines level in hypothalamus. So increase in catecholamines level will increase prolactin inhibiting factor and vice versa. Also drugs as phenothiazine and reserpine increase prolactin secretion through decrease of catecholamine levels Guyton, (1981).

Within 30 minutes of the onset of suckling, the prolactin level rises and after about three hours it falls. Frequent feeding which is the role in many "traditional societies" may explain the association between prolactin levels and successful prolonged breast feeding (Findlay, 1974).

In nursing mother Tyson, (1977) has described three types of prolactin response to nursing these three types are as follow:

**Stage 1:**

Occurs during the first week postpartum. Prolactin levels are still elevated. Thus suckling causes only small increase.

**Stage II:**

Is observed from the second week to the third month postpartum, base line levels are two to three times normal following suckling.
Stage III:

From the third month until weaning, prolactin levels return back to normal and suckling produce no rise in prolactin levels.

Prolactin level rises in response to various psychogenic factors, stress, anesthesia, surgery, high serum osmolarity, exercise, nipple stimulation and sexual intercourses (Worthington and Roberts, 1985).

High levels of prolactin suppress ovulatory cycles. The contraceptive effect of breast feeding is dependent upon the maintenance of hyperprolactinemia by frequent feeding. This effect is of particular important in spacing of births in traditional societies (Rosa, 1974).

The mother can keep prolactin level high by:

The baby is attached effectively at the breast and is not given any artificial dummies or teats. The baby breast feeds as frequently as he wants usually every 1-3 hours and as long as he wants. The baby breast feeds at night when prolactin release in response to suckling is greatest (Royal College of Midwives, 1991).

(III) Glactogenesis:

An intact hypothalamic pituitary axis regulating prolactin and oxytocin is essential for the initiation and maintenance of lactation (Larson, 1978).
The sensory nerve ending located in the areola and nipple are stimulated by suckling. The afferent pathway is via the spinal cord to the mesencephalone and then to the hypothalamus leading to hypothalamic suppression of prolactin inhibiting factor secretion causing adenohypophyseal prolactin release which stimulates milk secretion (Williams, 1981).

Oxytocin is responsible for the affection of milk. It is secreted by the posterior pituitary as a result of stimulation of mechanoreceptors of the nipple. Oxytocin is secreted into the blood and acts on the uterus and the breast leading to an increase in blood flow and arise in skin temperature (Lind et al., 1971).

The most important action is on the myoepithelial cells which surround the alveoli resulting in milk being squeezed out and ejected into the terminal lacteal ducts (Cowie, 1972).

Growth hormone may act with prolactin in initiating lactation but appears to be most important in maintaining established lactation. ACTH gradually increases during pregnancy, stimulates adrenals to secrete corticosteroids. High level is believed to be necessary for maintenance of lactation. Thyroxin appears to be important in maintaining lactation either through some direct effect on the mammary gland or by the control of metabolism. Thyrotropin releasing hormone (TRH) which stimulates the release of prolactin can be used to maintain established lactation (Worthington and Robert, 1985).
Estrogen affects the process of milk secretion probably by acting through the pituitary, the type of effect of estrogen however, is related to the level of estrogen in the blood. When the level is low as in virgin there is no prolactin secretion. If the blood level is suitable, as occurs in parturition, the pituitary gland can discharge prolactin (Harfouche, 1970).

Progesterone only minipills and other systems for releasing low dose of progesterone alone are widely used for contraception in breast feeding women around the world. There is good evidence to confirm their acceptability and their lack of effect on milk production (Froser, 1991).

Oxytocin release can be inhibited temporally by sever pain such as fissured, nipple, stress hormones resulting from doubt, embarrassment or anxiety, nicotine or alcohol. The mother can reduce this inhibition by relaxing and getting comfortable for feeds. Avoid embarrassing or stressful situations for feeds, expressing milk and gently stimulating the nipple and asking someone to massage upper back especially along the sides of the back (King, 1992).

Breast feeding reflexes:

Successful breast feeding depends on interaction between neonatal and maternal reflexes.
A- Neonatal reflexes :

1- Rooting reflex : 

The rooting reflex is the first to come into play. When infants smell milk they move their heads around attempting to find its source. If their cheeks is touched by a smooth object (Mother's breast) they will turn towards this object opening their mouth in anticipation of grasping the nipple (Rooting with their mouth for the nipple) (Barness, 1996).

2- Suckling reflex :

The contact of the nipple against the palate and posterior tongue elicits suckling or "milking" and the buccal fat pads help to keep the nipple in place. This suckling reflex is a process of squeezing the sinuses of the areola rather than simply suction of the nipple (Barness 1996).

3- Swallowing reflex :

As the mouth of the baby becomes filled with milk. The baby swallows it reflexly (Jelliffe and Jelliffe, 1979).

b- Maternal reflexes :

There are three main maternal reflexes in the process of lactation :

1- Prolactin reflex "milk secretion reflex" :

Prolactin can be considered the key of lactogenic process initiating and maintaining milk secretion. Its production by the pituitary is mainly the result of prolactin reflex resulting from infant
suckling. Prolactin hormone in blood stimulates breast alveoli to secrete milk \textit{(Sulman, 1979)}. Prolactin reflex can be diminished by severe maternal malnutrition or hormonal response to the environmental and psychological stressful condition also, prolactin secretion and hence milk production is related to the amount of suckling stimulus \textit{(DeChateau et al., 1982)}.

The earlier and more frequently the newborn is allowed to suckle the breast the more the prolactin level and hence the more the milk produced \textit{(Winberg, 1986)}.

2- \textbf{Nipple erection reflex}:

stimulation of the mother’s nipple by the baby mouth leads to nipple erection making it more protractile. Failure of this protraction reflex is a common cause of difficulties in breast feeding \textit{(Eissa, 1988)}.

3- \textbf{Let-down reflex}:

Suckling also stimulates the release of oxytocin from the posterior lobe of the pituitary. oxytocin causes smooth muscle contraction throughout the body and particularly the myoepithelial cells of the breast. This results in regular emptying of the contents of the breast in baby's mouth or breast pump with successful let down reflex 90% of the available milk can be obtained by the baby in seven minutes or less \textit{(Guyton, 1981)}. Let down reflex can be facilitated or initiated by psychological factors, in some women it may be triggered as conditioned reflex responding to certain stimuli such as the sight or
sound of her baby or even the though of nursing, also it can be inhibited by the mother’s emotional stress and anxiety because in such conditions adrenaline is released causing constriction of blood vessels around alveoli preventing the circulating oxytocin from reaching its target organs (WHO 1982).
ADVANTAGES OF BREAST FEEDING

Breast feeding has many advantages for the baby, for the mother and for the community.

Anti-infective properties:

These anti-infective roles are mediated through:

I- Immunoglobulins and antibodies:

Human milk contains some immunoglobulin M (IgM) and little immunoglobulin G (IgG) but secretory immunoglobulin A (S-IgA) is the dominant antibody component (Kon, 1972).

S-IgA molecules resist proteolytic enzymes better than serum antibodies (Lindh, 1974) S-IgA antibodies do not activate complement, enhance phagocytosis, induce inflammation, they only bind to the antigen, be a bacterial, viral or food constituent in this way the S-IgA provides an antigen avoidance system (Soothill, 1993) preventing contact between the gut mucosa and potentially pathogenic microorganisms or offensive food (Hanson et al., 1985).
Table (I): Shows quantitative differences between mature human milk and Cow's milk in factors preventing infections

<table>
<thead>
<tr>
<th>Proteins (mg/ml)</th>
<th>Human</th>
<th>Cow milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactoferrin</td>
<td>1.5</td>
<td>Trace</td>
</tr>
<tr>
<td>Lysozyme</td>
<td>0.5</td>
<td>0.001</td>
</tr>
<tr>
<td>IgA</td>
<td>1.0</td>
<td>0.03</td>
</tr>
<tr>
<td>IgG</td>
<td>0.01</td>
<td>0.6</td>
</tr>
<tr>
<td>IgM</td>
<td>0.01</td>
<td>0.03</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cells (x/ml)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Macrophages</td>
<td>2000</td>
<td>0</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>800</td>
<td>0</td>
</tr>
</tbody>
</table>

Factors stimulating growth of lactobacilli

<table>
<thead>
<tr>
<th>Factor</th>
<th>Human</th>
<th>Cow milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.29</td>
<td>6.57</td>
</tr>
<tr>
<td>Buffering capacity (ratio)</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Titratable acidity (ratio)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Phosphorus (mg/L)</td>
<td>141</td>
<td>910</td>
</tr>
<tr>
<td>Lactose (gm/l)</td>
<td>71</td>
<td>47</td>
</tr>
<tr>
<td>Bifidus factor (ratio)</td>
<td>40</td>
<td>1</td>
</tr>
</tbody>
</table>

(Mc Laren and Burman 1982).

Breast feeding protect against intestinal, respiratory and allergic diseases (Peter et al., 1990)

S-IgA antibodies are acting against a wide variety of microorganisms and their product such as E-coli (Gruz et al., 1982) E-
coli heat toxin, vibrio cholera toxin (Holmgren et al., 1976) Shigella and Salmonella (Allardyce, 1974).

Rota virus antibodies (Simbon et al., 1979) and polio virus antibodies (Hanson et al., 1985) have been found in human milk. Gillin et al., 1983 reported that, human milk was lethal to Giardia lamblia and Entameba histolytica.

Juan et al. (1994) reported that the amount of anti giardia S-IgA in human milk was associated with prevention of symptoms of diarrhea due to giardia but not with acquisition of the organism.

A high degree anti viral activity against Japanese B-encephalitis and respiratory syncytial virus is present in breast fed infant (Downham, 1976).

2- Bifidus factor:

A specific factor demonstrated in human milk that supports the growth of lacto bacillus bifidus which metabolizes milk saccharides producing large amount of acetic acid, some formic acid and succinic acid that lead to a low pH of gastrointestinal contents of the breast fed infant and inhibits the growth of many pathogenic bacteria (Roberts et al., 1985).

The presence of bifidus factor is one reason why the stools of breast fed infants are different from those who are bottle fed (Cameron and Hofvander, 1983).
3- Lactoferrin:

Lactoferrin is a powerful bacteriostatic acting synergistically with S-IgA especially against pathogenic strains of E-coli as lactoferrin alone appears to have a slight inhibitory effect (Rogers and Synge, 1978) due to secretion by E-coli of iron chelator (Enterochlein) which ensures continuity of iron supply for the organism (Griffiths and Humphreys, 1977).

The bacteriostatic efficiency of the milk is at its maximum if the iron binding protein (Lactoferrin) is unsaturated (El-Behairy et al., 1979), so the use of supplement of oral iron in the breast fed infants may be contraindicated as interfering with protection afforded by lactoferrin at the same time iron supplementation to lactating women doesn't alter the bacteriostatic activity of breast milk lactoferrin on its degree of saturation (Reddy, 1977).

4- Complement:

C3 and C4 components of complement are known for their ability to fuse bacteria bound to specific antibody present in colostrum of human milk (Goldman and Smith, 1973).

5- Lactoperoxidase system:

It consists of lactoperoxidase, thiocyanate and hydrogen peroxide. It can inhibit or kill organisms such as Coliform, Salmonella, Shigella pseudomonous and Vibrios thiocyanate and the end product of oxidation of thiocyanate in the presence of lactoperoxidase and
hydrogen peroxidase have a damaging effect on the inner membrane of bacteria and can inhibit bacterial enzymes Rieter (1985).

6- Interferon:

Interferon consists of a family of secreted protein characterized by their ability to induce antiviral state in all cell types. Two types of interferon were recognized in human milk, type which is produced by macrophages and type which is produced by T-lymphocytes (Daniel et al., 1987).

Interferon increases the bactericidal capability of macrophages and also can either augment or suppress cellular and humoral immunity (Faltynek and Baglioni, 1984).

7- Binding protein:

Unsaturated B₁₂ binding protein of high molecular weight has been found in very high level in human milk. This binding protein renders the B₁₂ unavailable for bacterial growth of E-coli and bacteroids (Lawrence, 1985).

Cellular component of human milk:

Human milk especially colostrum contains large number of cell which decrease rapidly at the end of the first week, of these cells are:

a- macrophages:

Constitute about 80 % of total cellular content of colostrum. They phagocytose micro-organisms as bacteria and fungi. Also, they
help T-cell reactivation through cellular co-operation or by processing the antigen. Lawrence, (1985).

b- Lymphocytes:
Constitute about 10% of the total cell count of human milk 50% of which are T-lymphocytes. 34% are B-lymphocytes and the remaining are of non differentiated cellular type (Goldman and Smith, 1973).

All the above protection agents of human milk renders the breast fed infant less susceptible for many viral bacterial and fungal diseases than the bottle fed infant.

Human milk is considered a complete diet as it contains the essential elements for proper nutrition of infants and newborn including fat, protein, carbohydrate, vitamins and minerals.

1- Fat
The fat in human milk is ideally suited to infant digestion and absorption. It has a high content of oleic acid, linolic acid, phospholipids and inositol (Ogra and Green, 1982) The actual content of polyunsaturated fatty acids in human milk varies with maternal diet but it is generally between 8% and 24% of total fat content (Lammi keefe and Gensen, 1984).

Human milk triglycerides are structured with short and medium chain fatty acids in the alpha 1 and 3 positions ready for hydrolysis
and absorption. Saturated fatty acids predominate in the beta 1 or 2 position for enhanced fat digestion (Watkins, 1984).

Human milk also contains lipase which facilitates fat digestion (Chapple et al., 1985). Cow's milk because of its large proportion of short chain saturated fatty acids and the position of fatty acids on the triglyceride molecule is harder for an infant to digest. Arachidonic and docadexanic acids are present in higher concentration in human milk. This may be important as these substances may play a significant role in the biochemical development of the brain (Watkins, 1984).

2- Protein:

Human milk contains about 1.3 gm/dl which is about one third of the proportion content of cow's milk. The casein protein in human milk is much lower than that of cow's milk. The curd of the human milk is safe and flacculent in contrast with that of cow's milk which is tough and rubbery (Jellife and Jelliff, 1979).

Human milk is rich in essential amino acids required to help brain growth and their function in pre-terms and young full-term infants (Pholandt, 1984).

Also human milk is a good source of taurine which can't be synthesized by the new born but considered as an essential nutrient for them (WHO 1982).
Tyrosine and phenylalanine are lower in human milk than in the cow's milk. So it is suitable for young neonates and pre matures being have a limited ability to metabolize tyrosine and phenylalanine (Mamunes et al., 1976).

3- Carbohydrates:

Human milk contains the highest level of lactose among all, it contains about 7gm/dl. Lactose in human milk has many functions as it enhances absorption of calcium and so play a role in preventing rickets. lactose is metabolized in the intestine into glucose and galactose the latter is a constituent of glacto-lipid as cerebroides needed for the development of C.N.S., also it promotes the growth of lactobacillus in the intestine, Jellife and Jellife, 1979).

4- Vitamins:

Human milk contains all vitamins required for good nutrition and health (Wortheington et al, 1985)

Ziegler et al. (1985) found that the milk from mother with adequate vitamin D status will support the nursing infant's vitamin D requirements. Daily supplementation with 4001.u of vitamin D is recommended for breast fed infants when the quantity and/or quality of mother's diet is inadequate, or when the baby has negligible direct exposure to sunlight. For pre term infants daily supplementation of 400 to 600 vitamin D in advisable. Human milk contains higher level of vitamin A, ascorbic acid and vit. E than Cow's milk. (Chappel et al., 1986).
Vit K in human milk is lower than cow's milk but its absorption from human milk is facilitated by other nutrients such as calcium (D'conner, 1983).

Also, human milk contains significant amount of B-vitamins including riboflavin, thiamin, pantothenic acid, nicotinic acid, biotin vitamin B12 and folic acid (Ford et al., 1983).

5- Mineral and water :

Cow's milk contains much more of all minerals except iron and copper than human milk with total mineral content of 7-7.75% in cow's milk and 1.5-2.25% in human milk (Barness, 1992).

Regarding calcium content it is proved that during the first 2 to 3 days of life the levels of calcium in breast milk fall significantly. The higher calcium in cow's milk is paradoxically associated with a much greater incidence of neonatal hypocalcemia (Oppe and Redstone, 1986) due its lower bioavailability while there is evidence of excellent calcium absorption in infants fed human milk.

The efficient absorption of calcium from human milk is due to many factors including the high lactose content, the composition and structure of the fat and low buffering capacity (American Academy of Pediatrics, 1978).

However the most important factor is the degree of mineralization of the skeleton and the effect being mediated by feed
back control of the synthesis of 1,25 OH Cholecalciferol (Deluca, 1978).

For a very long time the iron content of human milk was thought to be too low and this was often used as an argument in favour of early feeding of other feeds so that iron deficiency would be prevented. But it was found that the iron in human milk is highly bioavailable and well able to satisfy the exclusively breast fed infants need for the first six months, (UNICEF and WHO, 1993). The low iron level and the form of iron in human milk plays also an important role in concert with specific anti-infective factors in protection against infection as lactoferrin and transferrin (Lawrence, 1985).

Trace elements:
Manganese and copper in human milk are much higher than in cow's milk (Atkinson et al., 1980)

Anti-allergic properties of human milk:
The incidence of cow's milk sensitivity is probably 1% to 2% during the first 2 years of life (Foucard, 1985).

During the neonatal period the small intestine has increased permeability to macromolecules. Proteins of breast milk are species specific and therefore non allergic for human infant, it has also been shown that macromolecules in breast milk are not absorbed (Lawrence, 1985).
Allergy and intolerance to cow's milk are responsible for significant disturbances and feeding difficulties not seen in breast fed infants (Bareness, 1992).

S-IgA in colostrum and breast milk prevents the absorption of foreign macromolecules when infants system is immature. S-IgA also promotes closure of the gut and therefore decreases the permeability of allergen's (Lawrence, 1985).

Health benefits to the mother:

Oxytocin that is released while breast feeding contract the uterus and helps to stop bleeding after delivery. This makes it important that the breast feeding must begin immediately after birth and continue frequently (Unicef/WHO, 1993).

Breast feeding is a strong natural contraceptive at the population level although frequent and prolonged breast feeding is required for full effect (Hanson et al., 1985).

Breast feeding reduces the probability of conception because it extends the postpartum anovulatory period and reduces the likelihood of conception once ovulation has occurred (Huffman, 1986).

Suckling frequency has been shown to be a major determinant of the probability of ovulation. Suckling frequency of more than five times per day with a total duration of more than 35 minutes and a
minimum of 10 minutes per feed was sufficient to maintain complete suppression of ovarian activity (*McNeilly et al.*, 1985).

The practice of night feeds has been shown to be particularly important in maintaining an ovulatory cycle (*Gross and Easterman*, 1985).

**Emotional satisfaction:**

The psychogenic advantages of breast feeding for both infant and mother are well recognized and successful breast feeding is a satisfying experience of both. Breast feeding mother gains a feeling of being essential and sense of accomplishment. The infant gains a close and comfortable physical relationship with mother (*Barness*, 1992).

**Other benefits for the baby:**

A lower of cot death (sudden infant death syndrome). An infant is recognized to have died from sudden infant death syndrome after the rough examination fails to demonstrate any other cause for death. Many hypotheses have been proposed to explain it, some deficiencies/problems are related to the infant defect in sleep breathing control, severe botulism infection and hyper sensitivity. Most of these circumstances can be associated with a lack of breast feeding (*Bernshow*, 1991).

There are also a lower incidence of risk of childhood diabetes and ear infection, orthopedic and dental problems, better psychomotor, emotional and social development (*Unicef / WHO*, 1993).
Economic value of breast feeding:

Breast feeding cause no burden on the income of the family or the community. Economically it is time saving. Available and ready at the proper temperature at any time the baby needs, also breast milk needs no sterilization or preparation. Many evidences show that, infant mortality and morbidity are much lower in breast feeding than in bottle feeding infants (McCann et al., 1984).

Breast feeding is protective against dental caries which is common in bottle be feed infants (Jelliffe and Jelliffe, 1979).
LACTIC ACID SYSTEM

The high-energy phosphates must continually be resynthesized at a rapid rate for strenuous exercise to continue beyond a brief time period. In such intense exercise the energy to phosphorylate ADP comes mainly from glucose and stored glycogen during the anaerobic process of glycolysis with the resulting formation of lactic acid. In a way this mechanism of lactic acid formation “buys time”. It allows for the rapid formation of ATP by substrate phosphorylation even, though the oxygen supply is inadequate on the energy demands outstrip the capacity for ATP resynthesis aerobically. This anaerobic energy for ATP resynthesis can be though of as reserve fuel that is brought into use by the athlete (Kicking) the last portion of a mile run. It is also of critical importance in supplying the rapid energy above that available from the stored phosphagens during a 440 yard run or 100 yard (Alpert, 1985).

The most rapidly accumulated and highest lactic acid levels are reached during exercise that can be sustained for 60 to 180 seconds. As the intensity of all out exercise decreases a corresponding decrease in both the rate of build up and final level of lactic acid (Karlesson, 1981).

Lactate Accumulation:

Lactic acid doesn't necessarily accumulate at all levels of exercise. During light and moderate exercise the demands of both groups are adequately met by reactions that use oxygen. In
biochemical terms, the ATP for muscular contractions made available predominantly through energy generated by the oxidation of hydrogen. Any lactic acid formed in light exercise is rapidly oxidized. As such the blood lactic acid level remains fairly stable even though oxygen consumption increase (Costill et al., 1973).

Lactic acid begins to accumulate and rise in an exponential fashion at about 55% of the healthy untrained subject's maximal capacity for aerobic metabolism (Hagberg, 1980).

The usual explanation for the increase in lactic acid is based on the assumption of relative tissue hypoxia in heavy exercise. It is argued that under these conditions of oxygen deficiency the energy requirement is partially met by a predominance of anaerobic glycolysis as the release of hydrogen begin to exceed its oxidation down, the respiratory chain consequently excess hydrogen are passed to pyruvic acid and lactic acid accumulates (Katz and Sahlin, 1990).

This increase in lactic acid becomes greater as exercise becomes more intense and muscle cells can't meet the additional energy demands aerobically (Costill, 1970).

This pattern is essentially similar for the trained subjects except that the threshold for lactate build up, termed the anaerobic threshold, or more precisely blood lactate threshold, occurs at a higher percentage of athlete's aerobic capacity (Gladden, 1989).
This favorable response could be due to the endurance athlete's genetic endowment or specific local adaptations with training that would four the production of less lactic acid (*Weltman, 1989*).

As well as a more rapid rate of removal at any particular exercise level (*Donovan and Brook, 1983*).

For example it is documented that capillary density as well as the size and number of mitochondria increase with endurance as does the concentration of various transfer agents involved in aerobic metabolism (*Young et al., 1983*). Such alteration certainly enhance the cell's capacity to generate ATP aerobically especially through the breakdown of fatty acids, and may extend the percentage of one is maximum that can be sustained before the onset of blood lactate accumulation (*Sjodin et al., 1982*). Trained endurance athletes, for example, perform at exercise intensities that represent about 80 to 90% of their maximum capability for aerobic metabolism (*Wasserman, et al., 1981*).

It is also suggested that lactate formed in one part of a working muscle can be oxidized by other fibers in the same muscle or by less active neighboring muscle tissue (*Jacobs et al., 1987*).

These adjustments and training adaptations would certainly help to keep lactate levels low during exercise and would also provide an important means for glucose conservation in prolonged work (*Coggan et al., 1990*).
Lactate - producing capacity:

The ability to generate a high lactic acid level in all out exercise in increased with specific anaerobic training and subsequently reduced with detraining. Well trained athletes have shown that when they perform maximal short-term exercise, the blood lactate level is 20 to 30% higher than in untrained subjects under similar circumstances. (Gladden, 1989). The mechanism for this response in unknown, but it may be due to large difference in motivation level accompanying the trained state as well as about 20% increase in enzymes involved in glycolysis, specifically phosphofructokinase observed as a result of anaerobic type training. (Tan et al., 1984).

Because lactic acid is continuously removed during and after exercise at a varied rate among individuals it is unlikely that blood lactate measured at a particular time in recovery gives the full picture of an individual's capacity for an aerobic metabolism (Alpert, 1985).

It is also likely that the increased intramuscular glycogen stores that accompany the trained state allow for a greater contribution of energy via anaerobic glycolysis (Tan et al., 1984). Although increase in enzymes of the anaerobic pathway have been reported with sprint type training (Fournier, 1982).

These changes do not appear as impressive as the changes in aerobic enzymes with endurance training (Holloszy and Coyle, 1984).
BREAST FEEDING PROBLEMS

A. Maternal Problems:
1- Retracted Nipple:

Retracted nipple or non-projectile nipple is difficult to be grasped by the baby. So, proper antenatal care of the nipple by massage and manual traction is important and in some cases breast pump or shield may be helpful (El-Mougi, 1984).

2- Engorgement:

A common problem facing nursing mothers usually starts from the second to the fifth day post-partum due to infrequent nursing or impaired let-down reflex (Jones, 1983).

During engorgement, the nipple areolar junction becomes firm, retracted and difficult to be grasped which may lead to sore nipple (Neifert and Seacat, 1986).

Unrelieved engorgement rapidly results in involution of mammary glandular tissue with subsequent scantily milk secretion. Also, it may be complicated by mastitis, sore nipple or breast abscess (Fleet and Peaker, 1978).

So, breast engorgement should be avoided by the mother and the baby should not be separated in early weeks after birth since breast fills more rapidly at this time. Also, frequent nursing day and night as early as the mother condition allows together with massage, manual
expression and hot compresses is helpful (Royal College of Midwives, 1991).

3- Sore nipples:

Sore nipples are common, transient and fairly mild problem, but remain a prevailing cause of early termination of breast feeding among women who had not anticipated discomfort with nursing. Soreness usually begins on the second or third day and is more pronounced at the beginning of feeding. It can be exacerbated by inappropriate technique or impaired let down reflex (Neifert and Seacat, 1986).

Causes of sore nipple:

Poor attachment, the mother's breast become engorged because her baby may not breast fed frequently, the baby has not established an effective suckling pattern, the baby has a thrush infection which has been passed to the mother's nipple and the baby in tongue tied because of a short frenulum to the extent that the tongue can not extend over the lower lip (King, 1992).

Management of sore nipple:

Must check the baby's position at the breast and improve it if necessary, this may be all that is needed, or apply expressed breast milk to the mother's nipple after a breast fed to lubricate and soothe the nipple tissue, apply warm wet cloth to the breast before the feed to stimulate let down, begin each breast feed on the least sore breast, avoid the limitation on the frequency of feeds, if the baby has fallen asleep at the breast and is no longer actively feeding but remain
attached, gently remove the baby from the breast, expose the breast to air and sun aid in healing, treat the thrush both on the mother's nipple and in baby's mouth, and lastly if the baby's frenulum is so short that the tongue can not extend over the lower lip and the mother's nipples have been sore for two or three weeks, consider if the frenulum should be clipped "this is rarely needed" (Unicef/WHO, 1993).

4- Thrush infection:

Source of thrush infection coming out of the birth canal, a baby may contract the oral thrush on infection caused by the yeast candida albicans. The baby may then transmit the oral infection to the mother's breast. It is important to treat both the mother and the baby (Rentrew et al., 1990).

Sings of thrush infection:

The mother's nipple may look normal or red and irritated there is deep penetrating pain and the mother may state that her nipple "burn and sting" after a feed.

The nipple remain sore for a prolonged time despite correct attachment, this may be the only sign of infection, the baby may have a white patches on the mucosa of his mouth and may have a fungal diaper rash and the mother may have a vaginal yeast infection (Unicef/WHO, 1993).
Treatment of the mother:

Apply medication to the nipple, use a light film of 1/2 - 1/4% gentian violet once daily for 3 days or apply nystatin or other antifungal cream after every feed until the infection is cleared. It is not necessary to wash these before a breast feed, air dry and expose the nipples to sunlight after each breast feed. If breast pads are worn replace them when they became moist, wash hands well after changing the baby's diapers and after using the toilet and if a vaginal infection is present treat it according to national protocols, the woman's partner may need to be treated also (Royal College of Midwives, 1991).

Treatment of the baby:

Apply 1/2 - 1/4% gentian violet after a feed every other day until the symptoms are gone for 3 days or apply nystatin drops after every feed for 14 days. The mother should wash her hands well and use a cotton swab or finger to apply the medicine into the corners of the baby's mouth including cheeks, gums and tongue. If the baby has a diaper rash, apply gentian violet once every other day or nystatin cream 4 times day to the diaper area until the symptoms have been gone for 3 days, and stop the use of any dummies, pacifiers, teats or nipple shields if they are used, they must be boiled for 20 minutes daily and replaced weekly (Lawrence, 1985).

5- Mastitis:

It is a clinical and pathological term which describe wide range of inflammatory disorder of the breast beginning with bacterial
cellulitis and it is usually secondary to inadequate nursing, milk stasis, and nipple fissure \textit{(Worthington - Roberts and Taylor, 1985)}.

Early treatment of mastitis with 10 days course of antibiotics is important to prevent abscess formation. Continued breast feeding is recommended, no ill effects have been shown in nursing infant continued breast feeding shortens the course of illness in the mother and decrease risk of abscess formation \textit{(Marshal et al., 1975)}.

6- Breast abscess:

Breast abscess formation is a major complication of postpartum mastitis, often associated with abrupt weaning during the illness or with delay in initiation of antibiotics \textit{(Marshal et al., 1975)}. While breast feeding is continued on unaffected side, the affected breast should be temporarily emptied by manual or gentle pump expression preferably in conjunction with let down reflex \textit{(Devereux, 1980)}.

7- Breast rejection:

Infants have been observed to reject the breast intermittently most often at 3 to 4 months and then go back after several feedings or a day. Total rejection of both breasts may be due to return of menstruation. In unilateral breast rejection, manual expression or softening the nipple for easier grasp may help. Holding the infant in some positions i.e., on same side in same direction "foot ball hold" for other breast may lead infant to take second breast \textit{(Lawrence, 1985)}.
8- Nipple confusion:

Infant may have difficulty in grasping the mother's soft, relatively flaccid nipple, usually following early infant exposure to artificial nipples and pacifiers. The confusion arises from the different tongue and mouth action required by each type of nipple, with breast feeding requiring a much more active role on the part of the infant. The failure to suckle effectively soon leads to mammary involution and diminishing milk supply (Neifert and Seacat, 1986).

B- Infant problems:

Babies who "refuse" the breast in the early days, may seem reluctant to breast feed. This is frequently because of something unrelated to feeding, it may simply be that the mother and the baby need time to learn how to breast feed (Unicef/WHO, 1993).

1- The baby who resists going to the breast:

When moved toward the breast, instead of starting to suckle, the baby's cries loudly. He may seem to fight against being brought to the breast. The more the mother tries, the more the baby cries (King, 1992).

Possible causes:

Someone "the mother or health worker" is pushing the back of the baby's head in an attempt to get him to attach. The baby was given artificial teats or pacifiers & dummies which have resulted in suckling confusion.
Management: Help the mother to hold her baby calmly, close to the breast. If the baby starts to cry or fight the breast, stop and try again about a minute after the baby is calmed, don't use artificial teats, pacifiers and dummies, don't put pressure on any potentially painful site, avoid holding the baby in "a feeding position" when administrating a medical treatment, and feeding the baby expressed breast milk (EBM) in cup until he feeds at the breast (Lauwers, Woessner, 1989).

2- The baby who can not get attached:

The baby seems to be hungry, but when he brings his mouth to the breast, he can not attach and possible causes include, the baby must twist his neck to breast feed, the baby does not open his mouth large enough, the baby has been given an artificial teat and developed suckling confusion, he may thrust of hump his tongue when he tries to attach and suckle, and the mother's nipples are inverted to the point that the baby can not get attached (UNICEF/WHO, 1993).

Management:

Be sure that the mother is holding her baby correctly leave the baby with the nipple until he opens his mouth wide before attaching, give no artificial teat to the baby and all suckling on the breast only, express milk from engorged breast and drowout on inverted nipple (Lawarence, 1985).
3- The baby who can not stay attached:

The baby attaches and begins feed, after a short time he falls away from the breast and cries possible causes include, the baby is unable to breathe when he is at the breast, the mother is moving either her breast or her baby or not supporting the baby enough so that the breast falls away, and the mother’s milk is flowing too forcefully (King, 1992).

Management:

Be sure that the mother is holding her baby correctly, don’t flex the baby’s head forward so that his nose is pushed against the breast, hold the baby in a side sitting position with head cradled in the mother’s hand for great head control, be sure that the mother’s breast doesn’t became too full, and suggest that the mother express milk before a feed (Unicef and WHO, 1993).

4- The baby who does not suckle:

The baby attaches to the breast but is not suckling and possible causes include, the baby is sleepy, the baby is not hungry, the baby is weak because of low weight gain and the baby is ill (King, 1992).

Management:

Don’t give unneeded drugs to breast feeding mothers to prevent sedation of the baby, give no artificial feeds, water or infant formula, make sure that the baby has enough breast in his mouth, and express milk to maintain lactation. Cup feed with EBM to increase the baby’s weight, energy, and health (Lauwers and Woessner, 1989).
5- The baby who refuses one breast:

The baby feeds well on one breast but refuses or does poorly when he is offered to other breast, and possible causes include, there is a difference in the mother's nipples or milk flow from each breast the mother is better at attaching her baby to one breast than the other, one breast is more engorged and the tissue is firmer and the baby is in pain when he is held in a position to feed from the other breast (Lawrence 1985).

Management:

Evaluate the mother's breast to note any difference that might be interfering with the baby's effort to achieve attachment (e.g. inverted nipple or difference in shape or size), assess the baby's position and attachment of both, express breast milk to maintain lactation both breasts, feed the EBM to the baby with a cup, and allow the baby to feed on only one breast if he continues to refuse the other breast, if he is allowed to feed when he wants, he will get enough milk from one breast alone (King, 1992).
VARIABLES AFFECTING BREAST MILK

Human milk is a highly variable product and its composition is known to be altered by several factors including maternal diet, maternal age, maternal nutritional status, stage of lactation and infant’s demand for milk (Pereira and Barbosa, 1986).

There are many factors that interfere with and affect breast feeding, of which some are related to the mother and others are related to the infant.

Maternal factors include:
1- Maternal nutrition:

Mother’s diet before and during pregnancy as well as lactation is very important as it can affect her offspring through fetal stores and low birth weight, through inadequate weight gain in pregnancy which act as an energy store providing breast milk with 300 kcal daily for 3-4 months (Hyttén and Leitch, 1987).

Maternal malnutrition also can affect composition and volume of breast milk produced but in a slight to moderate degree. Maternal stress can inhibit the let-down reflex, if there is loss of confidence or feeling that the baby does not get sufficient food even though no signs of underfeeding are obvious (Jelliffe and Jelliffe, 1979).
2- Maternal employment and education:

The working mothers reduce the number of breast feedings per day and at the time increase the supplementary feedings for day, accordingly higher significant percentage of ovulation is noticed among them (Eshra et al., 1989).

The employed nursing mother may find that regularly scheduled breast pumping sessions can prevent uncomfortable fullness, eliminate potentially embarrassing leaking and obtain additional human milk for the baby (Auerbach and Guss, 1984).

Maternal education appears to have played a highly significant role in the health of the children as it is found to be the most significant variable inversely correlated with infections during the first year of life (Holmes et al., 1983).

3- Size and shape of the breast:

In some urban communities a woman may mistakenly thinks that breastfeeding will make her breast sag. It is reported that if there is a tendency to sag. This will be caused by the enlargement and reshaping of the breast during pregnancy but not during lactation (Cameron and Hofvander, 1983).

4- Maternal nutritional status:

Nutrition is intimately related to the cultural practices of communities (Richie, 1987) and three interacting factors play a major role, these are the economic status level of education and health status.
Of great importance is the implication of economic stress for adequate mothering as mothers will produce low weight infants, loosing many babies causing maternal exhaustion. Low income mother who delivered at home will immediately be involved in the pressures of family care and the tensions of urban life (Moodie, 1982).

Closely allied to poverty and hence to malnutrition is the problem over population (Gordon, 1989).

Factors related to the infant:

1- Oral anomalies:

May interfere with successful breast feeding, emptying of the breast and feeding the milk by a special nipple or through a gastric feeding tube may suffice until corrective measures are taken (Bahna and Heiner, 1985).

2- Premature and low birth weight infants:

Are actually the most in need of breast milk and the usual quantities of breast milk may not supply the infant with all of its needed protein, calcium, phosphorus, sodium, and iron (Forbes, 1986). The weak suckling reflex and lack of preparedness of the mammary glands may be attributed for decrease demand of infant to milk (Anderson, 1984). It is proved that in the first four weeks of lactation there is a higher concentration of calories, amino acids, sodium, chloride, and IgA and a lower concentration of lactose have been demonstrated in preterm milk as compared to mature milk (Polin and Fox, 1986).
3- Hyperbilirubinemia:

Breast milk jaundice may occur in breast fed infants few days after birth and persist for several days and occasionally weeks \textit{(Winfield and Mac Faul, 1979)} and this requires only temporary cessation of breast feeding.

4- Psychological factors:

From the infant's side, lack of somatosensory simulation occurring in differing degrees of separation from the mother in maternity nurseries means that the neonate is in some degree thrust from the security of the uterus into uncertain, unresponsive world which may possibly make more difficult the development of basic trust in human relationship later in life \textit{(Jelliffe and Jelliffe, 1979)}. 
SUBJECTS AND METHODS
SUBJECTS AND METHODS

This study had been carried out on 60 healthy lactating women between 2 and 6 months post partum and their healthy infants.

All subjects were randomly selected from those attending the vaccination clinic at Benha University Hospital. We classified them into 2 groups:

Group (1):

(Thirty mothers) with full breast.

Group (2):

(Thirty mothers) with empty breast (evacuate the breast before exercise).

Methods:

1- Good clinical examination and E.C.G.:

Were done for each woman before exercise and pretested questions were used include:
- Age, sex, wt, of both infants and mothers
- Residence of the mothers.
- Type of feeding whether breast alone or supplementary feeding was taken.

2- Graded Exercise test:

The mother performed severe exercise using motor driven treadmill the grade began at 0% and increased 2.5% every 2 minutes
until a voluntary maximal effort was achieved. Heart rate and BL.P were measured during exercise and recovery. The maximal H.R. was calculated from formula (220 - age (years) with S.D of 10 - 12 beats/minute (Brunwold, 1992).

3- Milk samples:

Were taken at rest before exercise and at 10,30 - 60 - 120 minutes post exercise. Milk was expressed by the mother by hand. 2 samples were taken at each time. One for lactic acid determination, the other for infant taste test. The baby was put to breast at each time after taking the samples in 30 mother.

4- Infant taste test:

Previously collected samples stored in refrigerator were warmed in a water bath to baby temperature. The samples were coded and presented randomly to the infant in a double design the mother presented the samples to the infant through a medicine dropper or directly. The infant was fasting 2 hours before the test. The infant acceptance was measured used by mothers perception.

5- The sample for lactic acid determination was frozied till assayed by the standard enzymatic technique (sigma chemical company).

**Principle:**

Lactate present in the sample is determined according to the following reaction:

\[ \text{Lactate} + \text{O}_2 \xrightarrow{\text{Lactateoxidase}} \text{Pyruvate} + \text{H}_2\text{O}_2 \]
H₂O₂ + 4-chlorophenol + 4 - a min oantipyrine → Peroxidase → Quinoneimine + 2H₂O + HCl

Reagents:

| Reagent 1 Standard | Lactate 3.00 mmol/l or 270 mg/l (27 mg/100 ml) | Sodium azide |

Concentration in the test:

<table>
<thead>
<tr>
<th>Reagent 2 buffer</th>
<th>PIPES buffer pH 6.8 100 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4-chlorophenol 5.4 mmol/l</td>
</tr>
<tr>
<td></td>
<td>surface - active agents 2 g/l</td>
</tr>
<tr>
<td></td>
<td>Sodium size 1 g/l</td>
</tr>
<tr>
<td>Reagent 3 Enzyme</td>
<td>4-aminoantipyrine 0.4 mmol/l</td>
</tr>
<tr>
<td></td>
<td>Peroxide ≥ 200 IU/l</td>
</tr>
<tr>
<td></td>
<td>Lactae oxidase ≥ 150 IU/l</td>
</tr>
</tbody>
</table>

Working solutions

We opened a bottle of Reagent 3 and discarded the rubber cap. We reconstituted the lyophilisation pellet with 10 ml of Reagent 2 and resealed bottle with a white screw capsule from the plastic sachet.

Stability:
- 2 weeks at 20-25°C
- 6 weeks at 2-8°C
- Wavelength 505 nm (492 - 550).
- Zero adjustment Reagent blank

<table>
<thead>
<tr>
<th></th>
<th>Reagent blank</th>
<th>Standard</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard (Reagent 1)</td>
<td>-</td>
<td>10 µl</td>
<td>-</td>
</tr>
<tr>
<td>Sample</td>
<td>-</td>
<td>- 1 ml</td>
<td>10 µl</td>
</tr>
<tr>
<td>Working solution</td>
<td>1 ml</td>
<td>1 ml</td>
<td></td>
</tr>
</tbody>
</table>

Mix.
Measure the absorbance after incubation for 5 min at 20 -25°C.

The color intensity is stable 1 hour

Linearity 10 mmol/l or 900 mg/l (90 mg/100ml)

\[
\text{Calculation} : \frac{A_{\text{sample}}}{A_{\text{standard}}} \times n
\]

\( n = \text{concentration of standard in mmol/l (or in mg/100ml)} \)

**Statistical design:**

The collected data were tabulated and analyzed using paired t test and student "t" test according to *Altman (1993)*, using computer with the microstat program.
Table (1): Distribution of the studied mothers according to residence.

<table>
<thead>
<tr>
<th>Residence St. group.</th>
<th>Rural</th>
<th>Urban</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Mothers with full breast (Group A)</td>
<td>15</td>
<td>50.0</td>
<td>15</td>
</tr>
<tr>
<td>Mothers with empty breast (Group B)</td>
<td>14</td>
<td>46.7</td>
<td>16</td>
</tr>
</tbody>
</table>

\[ X^2 = 0.067 \quad P > 0.05 \]

Table (1): Shows that no significant difference was found in distribution of mothers with full breast and mothers with empty breast as regard residence.

Table (2): Distribution of infants of studied groups according to sex.

<table>
<thead>
<tr>
<th>Sex St. group</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Infants of mothers with full breast</td>
<td>14</td>
<td>46.7</td>
<td>16</td>
</tr>
<tr>
<td>Infants of mother with empty breast</td>
<td>17</td>
<td>56.7</td>
<td>13</td>
</tr>
</tbody>
</table>

\[ X^2 = 0.601 \quad P > 0.05 \]

Table (2) reveals that male infants of mothers with full breast and with empty breast were (46.7% and 56.7%) and also female
infants were (53.3% and 43.3%). The difference is statistically insignificant ($P > 0.05$).

**Table (3):** Means and standard deviations of some sociodemographic parameters of the two studied groups of mothers.

<table>
<thead>
<tr>
<th>Diff. Parameters</th>
<th>Mothers with full breast ($n = 30$) $\bar{X} \pm SD$</th>
<th>Mothers with empty breast ($n = 30$) $\bar{X} \pm SD$</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>27.13 ± 4.19</td>
<td>26.5 ± 4.23</td>
<td>0.58</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Wt (kgm)</td>
<td>70.97 ± 5.99</td>
<td>72.4 ± 7.49</td>
<td>0.82</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>161.5 ± 5.16</td>
<td>163.15 ± 6.15</td>
<td>0.96</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>* BMI (No. of children)</td>
<td>26.02 ± 1.94</td>
<td>27.29 ± 3.43</td>
<td>1.76</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td></td>
<td>2.6 ± 1.2</td>
<td>2.3 ± 1.4</td>
<td>0.88</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

* Body mass index = wt / (Ht)$^2$ meter

**Table (3):** Shows no statistically significant difference between $\bar{X} \pm SD$ according to age, height, BMI and no of infants among the mothers of the two studied groups.
Table (4): Means and standard deviations of some sociodemographic parameters of infants of the two studied groups.

<table>
<thead>
<tr>
<th>Diff. Parameters</th>
<th>Children of mothers with empty breast</th>
<th>Children of mothers with full breast</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months)</td>
<td>4.18 ± 1.24</td>
<td>4.2 ± 1.4</td>
<td>0.06</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Wt (kgm)</td>
<td>6.65 ± 1.09</td>
<td>6.12 ± 1.08</td>
<td>1.89</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>64.5 ± 3.6</td>
<td>63.03 ± 3.64</td>
<td>1.53</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>* BMI</td>
<td>15.87 ± 1.83</td>
<td>15.19 ± 2.01</td>
<td>1.37</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table (4): This table shows no statistically significant difference between $X \pm SD$ according to Age, Wt, Ht and B.M.I. among the infants of the two studied groups.
Table (5): Concentration of lactic acid in milk of mothers of both groups (with empty breast and with full breast) before and after exercise.

<table>
<thead>
<tr>
<th>St. group</th>
<th>Lactic acid in milk of mothers with empty-breast (n=30) X ± SD</th>
<th>Lactic acid in milk of mothers with full-breast (n = 30) X ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before exercise</td>
<td>0.72 ± 0.22</td>
<td>0.63 ± 0.15</td>
<td>1.8</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>10 min after</td>
<td>2.35 ± 0.35</td>
<td>2.44 ± 0.35</td>
<td>1</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>30 min. after</td>
<td>2.64 ± 0.33</td>
<td>2.87 ± 0.13</td>
<td>3.54</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>60 min after</td>
<td>1.39 ± 0.28</td>
<td>1.36 ± 0.39</td>
<td>0.34</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>120 min after</td>
<td>0.71 ± 0.45</td>
<td>0.69 ± 0.15</td>
<td>0.74</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table (5) and chart (1): Show that X ± SD of concentration of lactic acid in milk of mothers with empty breast is 2.64 ± 0.33 at 30 minutes while it is 2.87 ± 0.13 in milk of mothers with full breast. This difference is statistically significant (p < 0.01). Also, the table shows no significant difference in concentration of lactic acid at other times.
Table (6): Means and standard deviations of lactic acid in milk of mothers with empty breast according to acceptance by infants before and after exercise.

<table>
<thead>
<tr>
<th>Acceptance Time</th>
<th>Lactic acid in milk of mothers with empty breast</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Accepted $\bar{X} \pm SD$</td>
<td>Refused $\bar{X} \pm SD$</td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>0.7 ± 0.23 (n = 28)</td>
<td>74 ± 0.07 (n = 2)</td>
<td>0.61</td>
</tr>
<tr>
<td>- 10 min.</td>
<td>2.37 ± 0.37 (n = 27)</td>
<td>2.68 ± 0.08 (n = 3)</td>
<td>3.65</td>
</tr>
<tr>
<td>30 min.</td>
<td>2.56 ± 0.34 (n = 22)</td>
<td>2.86 ± 0.18 (n = 8)</td>
<td>3.31</td>
</tr>
<tr>
<td>60 min</td>
<td>1.39 ± 0.28 (n = 30)</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>120 min</td>
<td>0.7 ± 0.46 (n = 29)</td>
<td>0.8 ± 0.0 (n = 1)</td>
<td>1.12</td>
</tr>
</tbody>
</table>

Table (6) reveals that the concentration of lactic acid is higher in refused milk at 10 minutes ($2.68 \pm 0.08$) than that in accepted milk ($2.37 \pm 0.34$) this difference is statistically significant ($P < .01$).

Also lactic acid concentration is higher in refused milk at 30 minutes ($2.88 \pm 0.18$) than that of accepted milk ($2.56 \pm 0.34$).
This difference is statistically significant and no significant difference at other times (before exercise, at 60 minutes, at 120 minutes).

**Table (7):** Means and standard deviation of lactic acid in milk of mothers with full breast according to acceptance by infants before and after exercise.

<table>
<thead>
<tr>
<th>Acceptance Time</th>
<th>Lactic acid in milk of mothers with full breast</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Accepted $\bar{X} \pm SD$</td>
<td>Refused $\bar{X} \pm SD$</td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>0.63 ± 0.16 (n = 28)</td>
<td>0.71 ± 0.1 (n = 2)</td>
<td>1.04</td>
</tr>
<tr>
<td>- 10 min.</td>
<td>2.58 ± 0.27 (n = 25)</td>
<td>2.8 ± 0.18 (n = 5)</td>
<td>2.27</td>
</tr>
<tr>
<td>30 min.</td>
<td>2.8 ± 0.12 (n = 20)</td>
<td>2.98 ± 0.15 (n = 10)</td>
<td>3.3</td>
</tr>
<tr>
<td>60 min</td>
<td>1.36 ± 0.39 (n = 30)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>120 min.</td>
<td>0.68 ± 0.15 (n = 28)</td>
<td>0.75 ± 0.05 (n = 2)</td>
<td>1.56</td>
</tr>
</tbody>
</table>

Table (7): Reveals that the concentration of lactic acid is higher in refused milk at 10 minutes (2.8 ± 18) than that in accepted milk (2.58 ± 0.27). Also lactic acid concentration is higher in refused milk at 30 minutes (2.98 ± 0.15) than that in accepted milk (2.8 ± 0.12).
These differences is statistically significant.
No significant difference at other times.

**Table (8)**: Means and standard deviation of the difference of lactic acid concentration in milk of mothers with empty breast before and after exercises (n = 30).

<table>
<thead>
<tr>
<th>Time</th>
<th>Mean of the difference ± SD.</th>
<th>Paired t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 min.</td>
<td>1.65 ± 0.35</td>
<td>25.8</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>30 min.</td>
<td>1.92 ± 0.49</td>
<td>21.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>60 min.</td>
<td>0.7 ± 0.31</td>
<td>12.37</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>120 min.</td>
<td>0.31 ± 0.36</td>
<td>4.72</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**Table (8)**: Shows high significant difference (P < 0.001) of lactic acid concentration in milk of pre exercise and at 10 minutes, 30 minutes, 60 minutes and 120 minutes in milk of mothers with empty breast.
Table (9) : Means of the difference of lactic acid concentration in milk of mothers with full breast before and after exercise \((n = 30)\).

<table>
<thead>
<tr>
<th>Time</th>
<th>Mean of the difference ± SD</th>
<th>Paired t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 min.</td>
<td>1.82 ± 0.4</td>
<td>24.9</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>30 min.</td>
<td>2.22 ± 0.19</td>
<td>64.03</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>60 min.</td>
<td>0.73 ± 0.36</td>
<td>11.11</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>120 min.</td>
<td>0.12 ± 0.09</td>
<td>7.3</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Table (9) : Shows higher mean of the difference of lactic acid concentration in milk is \(2.22 \pm 0.19\) at 30 min. and this difference is highly significant \((P < 0.001)\) also shows high significant difference \((P < 0.001)\) of lactic acid concentration in milk of pre exercise and at 10, 60, 120 minutes in milk of mothers of full breast.
Table (10): Correlation coefficient between Lactic acid concentration in milk among studied mothers with empty breast pre and post - exercise.

<table>
<thead>
<tr>
<th>Time</th>
<th>Correlation coefficient “r”</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre - exercise</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Post - exercise</td>
<td>0.3566</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>10 min.</td>
<td>0.3638</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>30 min.</td>
<td>0.3935</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>60 min.</td>
<td>0.3361</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>120 min.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table (10) and chart (2) show significant correlation coefficient between concentration of lactic acid in milk pre and post exercise (P < 0.05) at different times except after 120 minutes in mothers with empty breast.
Chart (2) correlation coefficient between concentration of lactate acid in milk of mother with empty breast pre- and post-exercise.
**Table (11):** Correlation coefficient between lactic acid concentration in milk among studied mothers with full breast pre and post - exercise.

<table>
<thead>
<tr>
<th>Time</th>
<th>Correlation coefficient “r”</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre - exercise</td>
<td>---</td>
<td>-----</td>
</tr>
<tr>
<td>Post - exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 min.</td>
<td>0.276</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>30 min.</td>
<td>0.3723</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>60 min.</td>
<td>0.3874</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>120 min.</td>
<td>0.0057</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Table (11) and chart (3) show significant correlation coefficient between concentration of lactic acid in milk pre and post - exercise except after 120 minutes in mothers with full breast.
Table (12): Infants acceptance and rejection of milk (empty breast mothers) according to methods of examination.

<table>
<thead>
<tr>
<th>Methods</th>
<th>Dropper (n = 15)</th>
<th>Breast (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>refused</td>
<td>Accept</td>
</tr>
<tr>
<td>Time</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Before</td>
<td>1</td>
<td>6.7</td>
</tr>
<tr>
<td>10 min.</td>
<td>2</td>
<td>13.3</td>
</tr>
<tr>
<td>30 min.</td>
<td>5</td>
<td>33.3</td>
</tr>
<tr>
<td>60 min.</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>120 min.</td>
<td>1</td>
<td>6.7</td>
</tr>
</tbody>
</table>

Table (13): Infants acceptance and rejection of milk (full breast) according to methods of examination.

<table>
<thead>
<tr>
<th>Methods</th>
<th>Dropper (n = 15)</th>
<th>Breast (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>refused</td>
<td>Accept</td>
</tr>
<tr>
<td>Time</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Before</td>
<td>1</td>
<td>6.7</td>
</tr>
<tr>
<td>10 min.</td>
<td>3</td>
<td>20.0</td>
</tr>
<tr>
<td>30 min.</td>
<td>6</td>
<td>40.0</td>
</tr>
<tr>
<td>60 min.</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>120 min.</td>
<td>1</td>
<td>6.7</td>
</tr>
</tbody>
</table>
DISCUSSION

The benefits of breast feeding for infant nutrition and health are universally recognized as breast milk is considered the ideal for infants and newborn supplying all required nutrients during the first four to six months of life (Pereira and Barbosa, 1986).

In a study made by Wallace and Rabin (1991) they found that a significant increase in lactic acid concentration in breast milk following maximal exercise and they noticed that infants of some mothers refuse to nurse or fuss during the post exercise feeding.

In a survey of lactating women who exercised of moderate intensity, 7% reported that their infants often had difficulty nursing following maternal exercise. It was postulated that there may be some byproduct of exercise that may affect the milk (Brown and Wallace, 1990).

The present study was carried out to compare infant acceptance of pre exercise and post exercise breast milk and to correlate the infant response to the lactic acid concentration in milk. It is carried out among the healthy lactating women between 2 and 6 months post partum and their healthy infants who attended the vaccination clinic at Benha University Hospital.

The age was 2-6 months as infants > 6 months of age cried and fussed because of method of milk presentation (the medicine dropper).
Infant younger than 6 months allowed the mother to present the milk without disturbance (*Farbaman, 1971*).

The study was carried out on 60 mothers and their infants. All parameters including (Residence, Age, Weight, Height) of mothers and infants were collected. Good clinical examination and ECG were done for each woman before exercise then the mothers, performed severe exercise using a motor tridmill. The milk samples were taken at rest, before exercise and at 10-30-60-120 min. post exercise. Infant taste test was done. The infant acceptance of the milk was measured by the mother perception. There was no significant difference in distribution of mothers with full breast and mothers with empty breast as regard residence table (1).

The percentage of male infants of mothers with full breast and empty breast was (40.7% and 36.7%) respectively and percentage of female infants was (53.3% and 43.3%). These differences were not significant (P > 0.05) Table (2).

In the present study there was no significant difference between $\bar{X}$ and $SD$ as regard age, height, BMI (wt/ht)² meter and number of infants of the two studied groups (Table 3,4).

As regard the concentration of lactic acid in milk of mothers of the studied groups we found that the concentration of lactic acid increase in the samples of 10, 30, 60 and 90 minutes with the peak
concentration at 30 minutes in mothers with full breast and those with empty breast.

The mean and S:D were $2.35 \pm 0.35 \text{ mmol/L}$ at 10 minutes in milk of mothers with empty breast and $2.44 \pm 35 \text{ mmol/L}$ in milk of mothers with full breast. Also the mean and S:D in milk of mothers with empty breast was $2.64 \pm 0.33 \text{ mmol/L}$ at 30 minutes and it was $2.87 \pm 0.13 \text{ mmol/L}$ with full breast. This difference was highly significant ($P < 0.01$) Table (5).

Our finding was in agreement with those of Wallace and Rabin (1991) as regard the concentration of lactic acid in breast milk following maximal exercise. They found that following maximal exercise, a significant increase in the concentration of lactic acid was found in the blood at 5 minutes post exercise and in breast milk at 10 minutes post exercise. The elevation of the lactic acid concentration at 30 minutes sample of breast milk was not significant different from resting samples. In their study seven active postpartum women (2 to 24 months) were exercised to a maximum on a treadmill. Blood was sampled via finger prick at rest, before exercise and at 5 minutes post exercise. Milk was collected via self expression at rest, before exercise and at 10 and 30 minutes post exercise. Both blood and milk were analyzed for lactic acid via enzymatic methods. They reached to the conclusion that maximal exercise can result in significant increase in lactic acid concentration in breast milk. However, our finding was not in agreement with this study as regard the time of maximal concentration. We found maximal concentration at 30 minutes in milk
of both studied groups. In our study we found higher lactic acid concentration in milk of mothers with full breast than in milk of mothers with empty breast. but the peak incidence in both the empty breast and full breast groups was at 30 minutes $\bar{X} \pm S.D$ was 2.64 $\pm$ 0.33 mmol/L and 2.87 $\pm$ 0.13 mmol/L Table (5).

Our finding was in agreement with the study of Ernsthauseen, et al. (1992) as regard the influence of the fullness of breasts on the concentration of lactic acid in post exercise breast milk. They found that the state of fullness of breast with milk is a factor which affects the concentration of lactic acid in breast milk following maximal exercise. They selected 23 lactating women classified to group E ($n = 11$) which nursed and/or evacuated their breasts before exercise and group F ($n = 12$) which did not nurse or evacuate their breasts at least two hours prior to maximal exercise. Milk was collected at rest, pre exercise and at 10, 30, 60 and 90 minutes post exercise and was analyzed for concentration of lactic acid.

This study demonstrated that:
1- significant increase in lactic acid in the milk at all post exercise collections for both groups.
2- Significant differences in the time of peak lactic acid concentration in milk (group F = 10 min. and group E = 30 min.).

In the present study we found that the concentration of lactic acid was higher in the refused milk at 10 min and 30 min in the milk of the two studied groups in the milk of the mothers with empty breast $\bar{X}$
± S.D at 10 minutes was \( 2.37 \pm 0.37 \text{ mmol/l} \) in the accepted milk compared to \( 2.68 \pm 0.08 \text{ mmol/l} \) in the refused milk. \( \overline{X} \pm \text{S.D} \) at 30 minutes was \( 2.56 \pm 0.34 \text{ mmol/l} \) in the accepted milk compared to \( 2.86 \pm 0.18 \text{ mmol/l} \) in the refused milk. In the milk of the mother with full breast \( \overline{X} \pm \text{S.D} \) at 10 minutes were \( 2.58 \pm 0.29 \text{ mmol/l} \) in the accepted milk compared to \( 2.8 \pm 0.18 \text{ mmol/l} \) in the refused milk. \( \overline{X} \pm \text{S.D} \) at 30 minutes were \( 2.8 \pm 0.17 \text{ mmol/l} \) in the accepted milk compared to \( 2.98 \pm 0.15 \text{ mmol/l} \) in the refused milk. These differences were statistically significant.

The usual explanation for the increase in lactic acid is based on the assumption of relative tissue hypoxia in heavy exercise. It is argued that under these conditions of oxygen deficiency the energy requirement is partially met by a predominance of anaerobic glycolysis as the release of hydrogen begin to exceed its oxidation down the respiratory chain consequently excess hydrogen are passed to pyruvic acid and lactic acid accumulates (Katz and Sahlin, 1990).

This refusal can be explained by the fact that the lactic acid can affect the taste of the milk and produces a sour taste that may be detected by the infant (Guyton, 1981). Seiner (1977), who documented gustofascial response (puckering fascial expression) to sour taste in infants as early as a few hours after birth. It is described as a low level reflex not involving cortical structures.

Paffmann et al. (1971), demonstrated that when two taste substances were mixed as in milk in which the predominant taste is
sweet because of high concentration of lactose, the intensity of the mixture is less than the intensity of the independent substances. On the other hand when one or particularly when both taste substances are weak the taste of some substance are enhanced rather than suppressed, in addition the sweetness of a substance decreased when acetic, lactic, citric acids are introduced and when PH is lowered.

*Kare (1971)*, demonstrated that taste buds found to be at the highest number in the 5 to 7 months old fetus. *Farberman et al. (1971)*, demonstrated that taste buds reach mature adult morphology at 13 - 15 weeks in utero and complete before birth. Therefore it is possible that lactic acid that accumulates in breast milk changes the taste of milk resulting in detection and sometimes rejection by the infant.

In our study we found that lactic acid concentration was high not only in the refused milk but also in the accepted milk. \( \bar{X} \pm S.D \) were \( 2.58 \pm 0.27 \) mmol/l and \( 2.8 \pm 0.12 \) mmol/l in the accepted milk of the mother with full breast at 10 and 30 min. respectively also the \( \bar{X} \pm S.D \) were \( 2.8 \pm 0.18 \) in the refused milk at 10 minutes the same concentration of the accepted milk at 30 minutes. The same was applied to the mothers with empty breast (Table 6-7).

This may be explained by the hypothesis that exercise not only increase lactic acid concentration in milk of lactating mothers but also increase other substances that may change the taste of the milk and further researches is needed to demonstrate it. In a study of *Lovelady et al. (1995)*, on the effect of exercise on plasma lipids and
metabolism of lactating women. They found that exercise increases high density lipoprotein, cholesterol levels but didn't affect other lipid concentration. Insulin response decreased as Vo2 max increased. There was no effect of time or group on glucose or thermic response. They select sedentary exclusively breast feeding women randomly assigned to an exercise (E) or control (c) group at 6-8 wk postpartum. E-subjects performed aerobic exercise 45 min/day for 5 days/wk. for 12 wk. Resting metabolic rate (RMR), energy expenditure, body composition and dietary intake were measured at 6-8, 12 - 14 and 18 - 20 weeks postpartum. Maximum oxygen uptake (Vo2 max) glucose and the thermic response and plasma levels were measured at 6-8 and 18 - 20 wk. Vo2 max increased by 25, Vs 5% in the E vs the C group respectively. (P < 0.0001). RMR was similar between groups and didn't change over time. Weight and percent body fat declined (P < 0.01) during the study but there was no difference between E and C groups. They found that exercise improves cardiovascular fitness during lactation but doesn't increase the rate of postpartum weight loss.

So there may be some products as cholesterol which may be increased in milk of lactating women following exercise and this with other factors may be responsible for the refusal of some babies to post exercise milk.

In a study of Altemus et al. (1995), on suppression of hypothalamic pituitary adrenal axis responses to stress in lactating women. They found that lactation suppressed a variety of physiological responses to stress. They investigated whether stress -
responsive neuro hormonal system are also restrained during breast feeding. They chose treadmill exercise as a stressor because this stimulus produces an exercise intensity dependent on activation of hypothalamic-pituitary adrenal axis and the sympatho medullary system that is independent on difference in physical conditioning among subjects. Ten lactating and 10 non lactating women between 7 and 18 weeks postpartum performed 20 min of gradual treadmill exercise. The final 5 min. of exercise was set to elicit 90% of the maximal oxygen uptake of each subject. Plasma ACTH, cortisol and glucose responses of exercise were significant attenuated in lactating women ($P < 0.0001$, $P < 0.05$ and $P < 0.001$ respectively) basal Norepinephrin levels were also reduced in lactating women ($P < 0.05$). These results indicate that stress responsive neuro hormonal systems are restrained in lactating women.

So the metabolism of the lactating women in response to exercise is different from the non lactating women. That may affect the composition of the milk.

This is not in agreement with the study of Prentica (1994), who found that no difference in body energy expenditure or resting metabolic rate following exercise. He examined the safety of recreational exercise for lactating mother in a prospective intervention study 33 women who were 6-8 weeks post partum and breast feeding were randomly chosen to join either exercise or control group. The exercise group took part in a program of aerobic activities averaging
4.5 sessions per week after 12 weeks, aerobic activities higher in the women who had exercised than in the controls.

In our study we found that high significant difference ($P < 0.0001$) of lactic acid concentration in milk of pre exercise and at 10 minutes, 30 minutes, 60 minutes and at 120 min. post exercise in milk of mothers of empty breast. The mean of difference was very high at 10 min and 30 minutes. It was $1.65 \pm 0.35 \text{ mmol/l}$ (paired $t$ 21.8 and $P < 0.001$) at 10 min and $1.92 \pm 0.49 \text{ mmol/l}$ (paired $t$ 25.5 and $P < 0.001$) at 30 minutes (Table 8).

The mean of the difference was higher in milk of mothers with full breast at 10 min and 30 min. It was at 10 min $1.82 \pm 0.4$ (Paired $t$ 24.9 $P < 0.001$) and at 30 minutes $2.22 \pm 0.19 \text{ mmol/l}$ (Paired $t$ 64.03 $P < 0.001$).

This was in agreement with the study of Ernsthauseen et al. (1992), they found that the fullness of milk in breast affect the concentration of lactic acid in post exercise breast milk.

In our study we find a significant correlation coefficient between concentration of lactic acid in milk pre and post exercise ($P < 0.05$) in both studied groups at different samples except the samples after 120 minutes Table (10-11). This mean that the concentration of lactic acid is still high at 60 minutes and decrease to about the level of normal at 120 minutes.
Our results was in agreement with the study of Janet et al. (1992), on infant acceptance of post exercise breast milk. They found that lactic acid concentration increase following maximal exercise at 10 min. and 30 min. and lactic acid remains elevated in milk for at least 90 minutes but the time in which lactic acid disappeared was unknown.

In our study there was no significant difference in the acceptance of milk as regard the method of administration of milk whether using medicine dropper or put to breast directly in the two studied group. This may be explained by the age of the infants. We choose age of infant between 2 and 6 months. Janet (1992) demonstrated that infant of 6 months of age cried and fussed because of method of milk presentation. Infants younger than 6 months allowed the mother to present the milk without disturbance.
SUMMARY
SUMMARY

The nutritional immunological, economic and psychological advantages of breast feeding are well recognized.

Wallace and Rabin (1991), found a significant increase in lactic acid concentration in breast milk following maximal exercise and they noticed that infants of some mothers refuse to nurse during the post exercise feeding.

Our study was carried out to compare infant acceptance of pre and post exercise breast milk and to correlate the infant response to the lactic acid concentration in milk.

It is carried out among the healthy lactating women between 2 and 6 months post partum and their healthy infants who attended the vaccination clinic at Benha University Hospital.

This study was carried out on 60 mothers and their infants. We classified them into 2 groups:

Group (1):

(thirty mothers) with full breast.

Group (2):

(thirty mothers) with empty breast (evacuate the breast before exercise).
Good clinical examination and all parameters including residence, age, weight, Height of the mothers and infants were collected. ECG was done for each women before exercise then the mothers performed severe exercise using a motor tridmill, the milk samples was taken at rest, before exercise, post exercise. Infant taste test were done. The infant acceptance of the milk was measured by the mother perception.

In our study we found that the concentration of lactic acid increased in the samples of 10,30,60 and 90 min. with the peak concentration at 30 min. in both the studied groups.

\[ \bar{X} \text{ and S.D. at } 30 \text{ min. were } 2.64 \pm 0.33 \text{ mmol/l in the group of empty breast and were } 2.87 \pm 0.13 \text{ mmol/l in the group with full breast. The difference was highly significant (P < 0.01). Also we found that lactic acid concentration was higher in milk of mothers with full breast than in milk of mothers with empty breast.} \]

Also we found that the concentration of lactic acid was higher in the refused milk at 10 min and 30 min in the milk of the two studied groups and the lactic acid concentration was high not only in the refused milk but also in the accepted milk so exercise may not only increase lactic acid concentration in milk of lactating mother but also increase other substances that may change the taste of the milk.
In our study we found a significant correlation between concentration of lactic acid in milk of pre and post exercise (P < 0.05) in both studied groups at different samples except at 120 min.

Also we found that lactic acid increased following maximal exercise at 10 - 30 - 90 min. and returned to about normal at 120 min.
CONCLUSION AND RECOMMENDATION

According to the previous results and observations we can conclude that:

1- Lactic acid concentration is increased following maximal exercise and the maximum concentration was at 30 min. post exercise.

2- The state of fullness of breast with milk was a factor which affects the concentration of lactic acid in breast milk following maximal exercise.

3- The lactic acid affected the taste of the milk and produced a sour taste that may be detected by the infant.

4- There is a significant correlation coefficient between concentration of lactic acid in milk pre and post exercise.

5- Lactic acid concentration returned to near the normal level at about 120 min.

We reach to the recommendation that the mother should nurse or collect milk for later feeding before exercising.

An alternative recommendation is to decrease the intensity of exercise below the lactate threshold to prevent the accumulation of lactic acid in milk.

The decision to alter nursing patterns or exercise patterns is a choice of the mother.
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الملخص العربي

مقدمة البحث:
لقد لاحظت بعض الأمهات المرضعات صعوبة إرضاع أطفالهن بعد التمرينات أو المجهود العضلي الشديد. وفي دراسة أجريت على الأمهات بعد المجهود العضلي المتوسط وجد أن 7% من أطفالهن يجدون نوعاً من الصعوبة في الرضاعة بعد إتمام التدريبات.

وقد أتعرض أن هناك منتج جانبي ينشأ في اللبن بعد المجهود العضلي ربما يؤثر على طعمه. وفي أبحاث سابقة وجد أن هذا المنتج هو حمض اللبنيك الذي يزيد بنسبة عالية في اللبن بعد التدريبات الشاقة، وقد لوحظ أن حمض اللبنيك له طعم حامضي يستطيع أن يتذوقه الطفل بعد الولادة مباشرة.

ولقد لوحظ أن الأطفال الرضع يفضلون الطعم الحلو عن الطعم اللاذع فتزداد معدل الرضاعة في المرحلة الأولى. وقد لوحظ تغيرات في ضربات القلب و معدل التنفس و حالة الرضاعة عندما يعطي للرضع نوع من الألبان الغير مألوف له أو المعتاد عليها.

الهدف من البحث:
أجرت هذه الدراسة لـ:
1- مقارنة مدى تقبل الطفل الرضيع للبن الثدي قبل وبعد قيام الأم بالتمرين الشديد.
2- محاولة الربط بين مدى إستجابة الطفل وتركيز حامض اللبنيك في لبن الثدي.

خطوات البحث:
قد أشتملت هذه الدراسة على 60 أم من الأصحاء وكذلك أطفالهن الرضع من عمر 6-7 شهور. ثم اختار هذه الحالات عشوائياً من الحالات المتردة على عبادة التعليمات في مستشفى بنها الجامعي.
وخضعت جميع الأمهات لما يلي:

1- فحص كامل ودقيق.

2- رسم قلب قبل إجراء التمرينات.

3- عمل تدريبات شديدة لكل أم بواسطة الجهاز الخاص لرسم القلب المجهود وتم ذلك تدريجياً حتى وصل إلى أعلى مجهود إختباري. وتم قياس معدل ضربات القلب.

4- عينة اللبن:

تم أخذ عينة لبن من الأمهات قبل التمرين وبعد 10، 30، 60 و 120 دقيقة من إجراء التمرين. وفي كل مرة تم أخذ عينات الأولى.

الثانية:

وضعت في التلاجة (20°C) لحين إجراء قياس لحامض اللبن.

5- إختبار التذوق للرضيع:

أخذت العينات من التلاجة وسخنت إلى درجة حرارة الغرفة ثم أعطت للطفل بدون ترتيب بواسطة قطارة. وتم إرضاع بعض الأطفال مباشرة من الثدي وتم منع الطفل من الرضاعة قبل إجراء الاختبار بساعتين. ولاحظت الأم مدى تقبل الطفل للبن واسطابه وذلك عن طريق مقياس خاص بذلك.
خلاصة النتائج

أوضح نتائج البحث أن:

1. تركيز حامض اللبنيك يزداد في اللبن بعد 10 دقائق من المجهود العضلي الشديد.

2. تركيز حامض اللبنيك يزداد بمرور الوقت.

3. تركيز حامض اللبنيك كان أعلى في اللين الذي تم تغذيه الطفل بعد 20 دقيقة و30 دقيقة من بداية التمرين عن تلك الذي تم تغذيه الطفل من هاتين الفترتين.

4. تركيز حامض اللبنيك كان أعلى في اللين الذي تم تغذية الطفل وذلك فانه يفترض أن هناك منتجات أخرى غير حامض اللبنيك مثل الكوليسترول من الممكن أن يزداد في اللين بعد المجهود العضلي الشديد.

وذلك يجب على الأم المرضعة إما أن ترضع الطفل قبل التمرين العضلي الشديد أو تقوم بجمع الرضاعة قبل بداية التمرين. البديل الآخر أن تقوم بتقليل درجة التمرين تحت مستوى إفراز حامض اللبنيك بدرجة تجعله لا يغير طعم اللبن.

القرار بتغيير طريقة الرضاعة أو درجة قوة المجهود العضلي هو اختيار الأم المرضعة.
مالة لنقل الطفلك من الثامنة بعضا قيام
الدماي بانتربين الشطب

فما مقيد من الملعب
أحمد عبد الفتاح عزب
بكالوريوس الطب والجراحة
توطنه للحصول على درجة الماجستير
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